after puberty); and poor outcome (further major surgery recommended).

Over 4–5 years, 44 patients fitting the study criteria were referred for further investigation. 42 patients underwent examination under anaesthesia and two requested outpatient examination. 37 patients were given routine referrals for adolescent genital assessment, and seven were referred for clinical reasons (two for clitoromegaly, two for abdominal pain, one for primary amenorrhoea, one for menorrhagia, and one for vaginal discharge). Mean age at examination was 15·1 (range 7·5–19·4) years. Most first surgical procedures were done during 1979–89 (range 1979–95). Mean follow-up time was 13·2 (4·3–19·3) years. Median age of first surgery was 0·6 (2 weeks–10·4 years) years. In two (5%) of 44 patients, major vaginal surgery was deferred until after puberty. In 42 (95%) of 44 patients, childhood genital surgery was intended to be definitive with seven (17%) of the 42 having additional surgery planned later in childhood but for most, 35 (83%) of the 42, the first procedure was intended to be single and definitive (26 of 35 were planned one-stage clitorovaginoplasties).

In 26 (59%) of the 44 patients the cosmetic appearance was good or satisfactory (table 2). Overall 15 (34%) had an acceptable outcome and 29 (66%) had a poor outcome. Analysing outcome by diagnostic group, 24% of Group A, 33% of Group B, and 55% of Group C had acceptable outcomes. Of those undergoing single-stage feminising genitoplasty, nine (35%) of 26 had already had undergone at least one further major genital surgical procedure before referral to our department. Of the remaining 17 (65%) of 26 patients, none would be able to use tampons without further treatment. Of these, three (18%) of 17 required dilators or minor surgery and 14 (82%) of 17 needed major surgery. Ten (26%) of 38 had undergone two or more clitoral procedures with one having undergone four clitoral reductions.

The outcomes of childhood genital surgery are substantially poorer than reported previously with nearly all children requiring further treatment. All surgery was done at specialist units and should give the best results available. This study is retrospective, but because of the numbers of patients involved, we believe it is representative. Nevertheless, despite planned routine referrals for all relevant adolescents, these patients may be those with the poorest outcomes. Additionally these children had surgery between 1979 and 1995. There have been changes in surgical techniques, equipment, sutures, and antibiotics since that time. More up-to-date procedures may have better outcomes, although there are few data to support this.

This study prompts a re-evaluation of cosmetic genital surgery in children. Most vaginal surgery can be deferred until after adolescence unless haematocolpos is a risk. Repeated clitoral surgery may be more damaging to sexual function than a single procedure. Clitoral regrowth occurred in 39% of patients. Children with mild clitoromegaly should have surgery deferred until they are old enough to be involved in the decision. Surgery should not damage genital sensitivity and sexual expression should be pleasurable with the ability for orgasm undiminished; there are currently no objective data for these outcomes. It is important that clinicians and parents understand that genital ambiguity cannot be corrected in infancy by a single procedure. For most individuals further treatment will be necessary in adolescence and the long-term impact of such treatment on adult sexual function is still unknown.

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Cold feet and prolonged sleep-onset latency in vasospastic syndrome

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People with vasospastic syndrome have cold hands and feet and abnormal vasoconstriction after local cold exposure. Normally there is a circadian rhythm of distal vasodilation, with onset in the early evening, which directly influences ability to fall asleep. We gave a sleep questionnaire to 32 patients with primary vasospastic syndrome and 31 healthy controls. People with vasospasticity had significantly prolonged sleep-onset latency both at onset of night-time sleep and after nocturnal disturbance. This prolonged latency could be associated with impaired capacity for distal vasodilation.

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An association between thermoregulatory processes and the initiation of sleep has long been recognised. The degree of
dilation of blood vessels in the skin of hands and feet, which increases heat loss at these extremities, is a good physiological predictor for the rapid onset of sleep. Some sleep disorders might be secondary to distal vasodilation failure. People with vasospastic syndrome often have cold hands and feet, and respond to cold stimuli or even emotional stress with inappropriate constriction or insufficiency dilation in the microcirculation. We studied whether this group of people have a specific sleep disturbance.

32 people with a primary vasospastic syndrome (three men and 29 women, median age 42 [IQR 24, range 21–76] years) and 31 healthy controls (four men and 27 women, median age 40 [IQR 27, range 18–77] years) were enrolled into the study. All participants were recruited from the University Eye Clinic, Basel, Switzerland. The controls consisted of companions of the patients, hospital staff not involved in the study, and patients referred for investigation of refraction. The distribution of age, sex, and occupational characteristics of the groups of participants were not significantly different. Vasospasm was defined as a combination of: positive medical history of cold hands or feet, and optionally a tendency towards low blood pressure, Raynaud’s phenomenon, migraine, tinnitus, and normal-tension glaucoma (NTG); and an abnormal result in the local cold exposure test in nailfold capillaroscopy. This test is done on individuals acclimatised at a 23°C room temperature, baseline measurements are then taken for 60 s, then a fingertip is subjected to carbon dioxide at −15°C for 60 s. People found to have a blood-flow standstill of at least 12 s in one or more capillaries were defined as having vasospasticity. A detailed medical history excluded individuals with a history of systemic disease (eg, diabetes, high concentrations of blood lipids, systemic circulatory diseases other than vasospasm, and restless leg syndrome) or who had been taking any systemic medication long-term. Because of a difficulty in accessing nailfold capillaries in two participants with typical vasospastic symptoms, they could not partake in the study. A detailed sleep questionnaire screened for individual sleep habits (duration, timing, and quality of sleep); specific items and open questions were added to assess possible reasons for difficulties in initiating or maintaining sleep.

The self-reported sleep characteristics of the control group fell within normal ranges. However, people with vasospasm rated their general quality of sleep to be worse more often than controls (35% vs 23%); they showed significantly prolonged sleep-onset latencies not only before nocturnal sleep (figure) but also after a nocturnal sleep interruption (median 15·0 [IQR 27·5] min vs 5·0 [IQR 8·0] min; p=0·004, Mann-Whitney U test) even though the number of sleep interruptions themselves did not differ between the groups. Moreover, 63% of the people with vasospasm named cold feet among their reasons for sleep onset difficulties, no one in the control group marked this item. Primary vasospastic syndrome is more common in females and in people with a low body mass index (BMI). In our study women predominated, and the BMI of the group with vasospasticity was lower than that of the control group (median 20·5 [IQR 3·7] vs 22·3 [5·2]).

Diagnosis of vasospastic syndrome can easily be made if there is a history of cold hands and feet, and noninvasive nailfold capillaroscopy combined with a cold provocation test can confirm diagnosis. Individuals with vasospastic syndrome are otherwise usually quite healthy, and vasospasm does not need special treatment. Vasospasm often involves ocular perfusion, and it has been shown to be a clear risk factor for glaucomatous damage, particularly in patients with NTG. In our study, we had 14 people with vasospasm and NTG and 18 people with vasospasm but without NTG; sleep onset latency (the main target variable) did not differ between the two groups (data not shown), allowing the combined analysis.

An explanation for a prolonged sleep-onset latency could be that people with vasospasm go to bed too early in relation to their endogenous circadian rhythm phase. Such a relative advance would indicate that the body physiology is not yet ready for sleep. Only a median 10 min advance in phase of the sleep episode compared with controls was found. A more precise study of circadian rhythm timing and amplitude will be required to follow up the possibility of circadian rhythm involvement. A rather selective self-reported sleep disturbance emerged in participants with vasospastic syndrome, indicating that the prolonged sleep-onset latency may be associated with impaired nocturnal distal vasodilation. Awareness of this connection may help in treatment by simple sleep aids such as a presleep warm bath, hot water bottle at the foot of the bed, or relaxation techniques. Also, it may be possible that sleep-onset latency is shortened by treatments already applied in vasospastic glaucoma patients—ie, magnesium, diprydiamole, and calcium-channel blockers.


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